UNIT NO. 5

COMMON INFECTIONS IN THE ELDERLY IN THE HOME OR NURSING HOME SETTING

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ABSTRACT

Infections in the elderly are associated with high morbidity and mortality. Diagnosing infections in the elderly is challenging due to their atypical and subtle presentation. A high index of suspicion is often needed. Commonly encountered infections in the elderly include bacterial pneumonia, urinary tract infection, cellulitis and Herpes zoster. In addition, institutionalised elderly and those with multiple hospital admissions are at risk of infection with Multidrug-resistant Organisms (MROs); this can be difficult to manage.[1] The purpose of this article is to look at some common infections in the elderly encountered in the home or nursing home, and review their management.

Keywords:
Elderly, Infections, Vaccination, Pneumonia, Urinary Tract Infection, Cellulitis, Influenza, Herpes Zoster, Antimicrobial Resistant Organisms.

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INTRODUCTION

Infections account for about 30% of deaths in the elderly.[2] The prevalence of pneumonia in the elderly is >2-fold that in adults and the prevalence of urinary tract infection is >20-fold that in adults.[1]

Symptoms in the elderly are typically non-specific and subtle. These include delirium, functional decline, anorexia or failure to thrive, falls, generalised weakness and exacerbation of chronic diseases such as congestive heart failure or COPD.[1,2] The home care patient may have considerable host risk factors which predispose them to getting infections. These include advanced age, multiple chronic comorbidities, functional decline, decreased physiologic reserves, defective protective mechanical barriers, and impaired cellular and humoral immunity.[1] In addition, they may have cognitive impairment or difficulty expressing themselves. Very often, the institutionalised elderly or those with repeated hospital admissions are also exposed to Multidrug-resistant Organisms (MROs).

In home care, diagnoses of infections are frequently made on an empiric basis based on the patient's symptoms and signs and history from caregiver. Cultures are not routinely obtained to diagnose infections of the respiratory or urinary tract unless systemic symptoms suggest septicaemia.[3] Given that the elderly also have a poorer body temperature response, any elevation of 1.1 degrees Celsius from their baseline should be considered a febrile response. Red flags that suggest more serious issues include: (1) temperature above 38.3 degrees Celsius or hypothermia; (2)tachypnoea; (3) vomiting; and (4) altered level of consciousness.[2]

Much of the care provided at home is less controlled than in a hospital environment. During visitation, commonly used nursing bags can also be a potential source of transmitting organisms, including MROs.[4] For the healthcare providers and caregivers, proper hand hygiene must be performed before and after contact with patient's skin and after contact with inanimate objects in the immediate vicinity of the patient. Patient care devices (e.g., blood pressure cuff, stethoscope) must be disinfected when visibly soiled and on a regular basis.[3,4] The use of protective equipment such as gowns, gloves and masks is recommended when dealing with body fluids or patients with MROs. Simple infection control strategies such as regular hand washing, wound care techniques and routine changing of urinary drainage bag on a monthly basis should be taught to the caregiver to minimise risk of infections.[3]

A. Pneumonia

Community-acquired pneumonia (CAP) refers to lower respiratory tract infections in patients who has not been hospitalised in the past 6 weeks. *Streptococcus pneumoniae* remains the most important cause of community-acquired pneumonia in the elderly. Other common aetiologic agents include *Staphylococcus aureus*, Gram-negative organisms, *Haemophilus influenzae*, respiratory viruses, *Pseudomonas aeruginosa*, *Legionella*, *Mycoplasma* and *Moraxella*.[5]

i. When to suspect?

The elderly are particularly prone to pneumonia because of their impaired gag reflex, impaired mucociliary function, reduced immunity and presence of multiple comorbidities.[5] Symptoms include new or worsening cough, newly purulent sputum, respiratory rate > 25 breaths per minute, tachycardia, new or worsening hypoxia, pleuritic chest pain, cognitive or functional decline, changes in respiratory exam, and fever (> 38.1°)or hypothermia (< 35.6°).

ii. How to assess the severity in the outpatient setting?

There are several assessment tools used to support clinical judgement. These include the Pneumonia Severity Index (PSI) and the CURB-65.[6] PSI uses 19 criteria including gender, age, chronic comorbidities, underlying disease, clinical signs, laboratory and radiology values to categorise patients into five risk classes. CURB-65 is much simpler to use and looks at Confusion, Urea, Respiratory rate, Blood pressure and Age >65 to predict mortality.[6] It does not require use of arterial

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blood gas measurements or radiology findings which are not readily available in the home setting.

Prognostic signs favouring hospitalisation in the elderly include the following:[6]
a. Nursing home resident;
b. Comorbid conditions (cancer, liver failure, congestive heart failure, renal failure, prior cerebrovascular accident);
c. Vital sign changes (Tachycardia >124 beats per min; tachypnoea >30 breaths per min; oxygen saturation <90% on room air or requiring >3L /min of O2 above baseline; hypotension with SBP <90mmHg or 20mmHg less than baseline; or temperature below 35º Celsius or above 40º Celsius);
d. Diagnostic changes include WBC <4000/mm3 or >130000/mm3; haematocrit <30%; arterial blood gas PaO2<60mmHg; or oxygen saturation <90%. Electrolytes BUN>29mg/dL, serum sodium <130mg/L and serum glucose >250mg/dL. Chest X-ray changes of multilobar infiltrates and/or pleural effusion; and/or
e. Presence of uncontrolled COPD or congestive heart failure or diabetes mellitus.

The mainstay of treatment for pneumonia is use of antibiotics and supportive care. Oral care, including cleaning the mouth after each meal, may help prevent bacterial respiratory infections from pharyngeal aspiration.

iii. Empiric therapy for mild cases/outpatient therapy
a. Beta lactam antibiotic with macrolide (PO Augmentin 625mg q8H with PO Clarithromycin 500mg q12H, or PO Azithromycin 500mg OD, or PO Moxifloxacin 400mg OD).
b. Fluoroquinolone (e.g., Moxifloxacin 400mg OD).
c. Suspected aspiration: Augmentin and Azithromycin, or Cephalosporin (e.g., Cefuroxime) and Azithromycin.

iv. Severe cases requiring hospitalisation: triple antibiotic coverage
a. Antibiotic 1: Broad-spectrum antibiotic with antipseudomonal coverage
(i) Ceftazidime IV 2g q8H;
(ii) Imipenem or meropenem IV 1g q8H; or
(iii) Piperacillin with tazobactam IV 4.5g q6H.

b. Antibiotic 2: Gram-negative and antipseudomonal coverage
(i) levofloxacin 750mg q24h;
(ii) IV ciprofloxacin 400mg q8H; or
(iii) aminoglycoside IV Amikacin 15-20mg/kg OD.

c. Antibiotic 3: MRSA coverage
(i) IV Vancomycin 15-20mg/kg q12H.

Generally patients with *Streptococcal pneumonia* should be treated for 7-10 days (3 days with Azithromycin) whilst patients with mycoplasma or chlamydia infection and those who are immunocompromised should receive longer regimes.

B. Healthcare Associated Pneumonia (HAP)

HAP should be suspected in any patient hospitalised for at least two days in the prior 90 days; who is a nursing-home or residential-care facility resident; or who is immunocompromised and has predisposing comorbidities (e.g., dialysis, chemotherapy, chronic wound, home intravenous antibiotics).[6]

Risk factors for being colonised and infected with MROs include the following:[7]
a. Antimicrobial therapy in prior 90 days;
b. High frequency of antibiotic resistance in the community;
c. Hospitalisation >2 days in the preceding 90 days;
d. Residence in nursing home or extended-care facility;
e. Home infusion therapy;
f. Dialysis;
g. Home wound care;
h. Previous infection with multidrug-resistant organism;
i. Having a family member with MRO; or
j. Being immunosuppressed.

In addition to the common organisms that cause pneumonia, additional organisms commonly involved in HAP include *Haemophilus influenzae, S. aureus, P. aeruginosa, Acinetobacter baumanii,* extended-spectrum beta lactamase producing gram-negative rods and MRSA.[7]

i. Treatment of HAP
For patients with no risk factors for infection with MROs, recommended therapy is:
a. IV Piperacillin-tazobactam 4.5g q6H.

If patients are at risk of acquiring MROs, add:
a. IV Vancomycin 15-20mg/kg q12H; with
b. Amikacin 15-20mg/kg once daily.

Cultures should be taken at day 2 or 3 of therapy and patient's clinical response assessed. If cultures are negative, antibiotics can be oralised to:
a. PO Ciprofloxacin 500mg q12H; with
b. PO Augmentin 625mg q8H.

Treatment should be given for a total duration of seven days, failing which if there is no clinical improvement and cultures are negative, testing should be done for other organisms such as Legionella, viruses or MROs, and a search for other sources of infection carried out.[7]

C. Influenza

Influenza is most commonly caused by 2 RNA viruses — Influenza A and Influenza B. The influenza B viruses are relatively stable but antigenic shifts in Influenza A viruses are responsible for influenza epidemics and pandemics.[8] Of deaths resulting from influenza, 80-90% occur in the elderly. The age-specific risks of influenza-related mortality increases

exponentially after age 65.[8] In tropical countries like Singapore, transmission and new cases occur throughout the year. Vaccination is the most cost-effective means to reduce mortality and morbidity associated with influenza.[2,9] Pneumococcal vaccination has been shown to reduce influenza-associated complications.[8]

i. When to suspect?

The clinical presentation ranges from a self-limiting upper respiratory tract infection to a severe illness with life-threatening complications. The natural course of the illness is determined by the virulence of the virus, the host immune system and presence of comorbidities.[8] Symptoms include sneezing, stuffy nose, headache, chills, muscle aches, malaise, cough and sore throat. The natural course is one week before recovery starts, although in the elderly weakness may persist a few weeks during which complications, including pneumonia, may occur. Pneumonia is a serious complication occurring in 5-38% of influenza cases and can result in hospitalisation and/or death.[8]

ii. Diagnosis

Influenza diagnostic tests do not need to be done on all patients. However, if the patient is frail, has multiple comorbidities, is immunocompromised or respiratory compromised, or requires hospitalisation, influenza diagnostic testing is recommended.[8] During a respiratory illness outbreak in hospital or nursing home, testing for influenza can be helpful to identify the strains.

Preferred samples are nasopharyngeal or nasal swab, and nasal wash or aspirate. Diagnostic tests available for influenza include viral culture, serology, rapid antigen testing, Polymerase Chain Reaction (PCR), immunofluorescence assays and rapid molecular assays. Viral cultures are essential for determining the influenza A subtypes and influenza A and B strains causing illness. Rapid tests are easy to perform and useful for ruling out the disease.[8] Rapid tests differ in the types of influenza viruses they can detect but cannot differentiate the subtypes. The main limitation lies in its lack of sensitivity in picking up a positive infection. Immunofluoresence assays and PCR are the tests of choice to diagnose influenza. PCR is highly sensitive and able to detect most types of other viruses, including rhinoviruses and coronaviruses.[8]

iii. Treatment

The goals of treatment are to alleviate and shorten the duration of symptoms; to prevent secondary bacterial pneumonia, primary influenza viral pneumonia, hospitalisation and death.[9] There are two main types of antivirals: M2 channel inhibitors such as Amantadine and Rimantidine; and neuraminidase inhibitors Oseltamivir and Zanamivir. They must be taken within 48 hours of onset of illness to be effective. The potential benefit of Oseltamivir is its ability to inhibit both Influenza A and B. It reduces the duration of symptoms by one to one and a half days. Oseltamivir should be taken at 75mg BD for 5/7 or OD if creatinine clearance is <30ml/min. Rimantidine and Amantadine inhibit Influenza A. Rimantidine has fewer side effects on the central nervous system in comparison. For elderly with normal renal function and documented Influenza A infection, the dose for Rimantidine is 100mg OD for 3-5/7. For creatinine clearance 20-30ml/min, the dose is 200mg twice weekly; and for creatinine clearance 10-20ml/min, 100mg 3x/week.[9]

iv. Prevention

Influenza vaccination is the most important tool for preventing morbidity and mortality from influenza amongst the elderly.[9] Those who are recommended to receive the vaccine include:[9]

a. Elderly who are >65 years old,
b. Individuals at risk of influenza and its complications (including those with chronic respiratory, cardiovascular, renal, liver or neurological disease);
c. People with diabetes or who are immunosuppressed; and/or
d. Institutionalised elderly.

Most of the inactivated vaccines contain two A strains and one B strain. Elderly persons may benefit from vaccination despite low antibody titres, as vaccination has been shown to reduce the risk of complications, hospitalisation and death.[9] In the elderly, it may take 4-6 weeks before optimum antibody titres develop. Common side effects for the influenza vaccine include local reactions such as erythema, induration, pain and warmth. There is no evidence for increased systemic side effects such as Guillain-Barre syndrome following vaccination.[8]

D. Urinary Tract Infection (UTI)

UTIs account for over a third of all nursing home associated infections.[9]

Risk factors include:

a. A history of previous UTI;
b. Post-menopausal women;
c. Benign prostatic hyperplasia with retention of urine;
d. Neurogenic bladder (in patients with Diabetes Mellitus, Alzheimer's and Parkinsonism);
e. Prolonged bed rest;
f. Urinary incontinence;
g. Faecal incontinence; and
h. Presence of indwelling catheter.[2,10]

Causative organisms include: *Escherichia coli* (most common), *Proteus mirabilis, Klebsiella spp, Enterobacter,* coagulase-negative *Staphylococcus aureus*, and polymicrobial urinary tract infections. Classical symptoms such as dysuria, fever, urinary frequency and suprapubic tenderness may be absent in the elderly.[2]

i. Asymptomatic bacteriuria

Over-diagnosis of UTI is common. It is important to differentiate between asymptomatic bacteriuria and urinary tract infection since the former does not require antibiotic

treatment. Urine odour or turbidity is not an indication to test urine and urine dipstick should not be routinely performed to detect asymptomatic bacteriuria.

Symptoms suggesting urinary tract infection include:
a. Dysuria;
b. Fever >37.9º or chills;
c. Frequency;
d. Urgency;
e. Flank pain;
f. Suprapubic pain;
g. Haematuria; or
h. Worsening mental or functional status.[11]

a. Diagnosis
Diagnosis of asymptomatic bacteriuria in females requires the presence of two consecutive urine specimens positive for the same bacteria strain in quantities $>10^5$ CFU/ml, in the absence of any signs or symptoms of a genitourinary tract infection. Asymptomatic bacteriuria in males requires a single void specimen with 1 bacterial isolate $>10^5$ CFU/ml in the absence of symptoms.[10,11]

Diagnosis of UTI in community-dwelling older adults requires the presence of genitourinary symptoms in the setting of urinary tract inflammation (pyuria) and a positive urine culture.

Diagnosis of UTI in institutionalised elderly requires the presence of genitourinary symptoms with a positive urine culture. However, chronic genitourinary symptoms such as incontinence, urgency and frequency, and dysuria may not always be communicated in the frail, institutionalised elderly. Instead, non-specific signs and symptoms such as anorexia, confusion and decline in functional status may be the presenting symptoms.[10,11] This makes diagnosing UTI in institutionalised elderly highly challenging.

According to the revised McGeer criteria, the diagnosis of UTI for institutionalised elderly without an in-dwelling catheter (IDC) includes[11]:

1. At least one of the following sub-criteria:
(i) Acute dysuria or acute pain, swelling or tenderness of testes, epididymis or prostate;
(ii) Fever (>38º Celsius) or leucocytosis with at least one of the localising urinary tract subcriteria: acute costovertebral pain or tenderness, suprapubic pain, gross haematuria, new or marked increase in incontinence, urgency or frequency; or
(iii) In the absence of fever or leucocytosis, then two or more of the following localising urinary tract subcriteria: suprapubic pain, gross haematuria, new or marked increase in incontinence, urgency or frequency.
2. One of the following microbiological sub-criteria:
(i) At least 10^5 CFU/ml of no more than two species of microorganisms in a voided urine sample; or
(ii) At least 10^2 of any number of organisms in a specimen collected by in- out catheterisation.

Finally, diagnosis of UTI in patients with IDC is defined by presence of symptoms or signs compatible with UTI with no other identified source of infection along with >103 CFU/ml of >1 bacteria species in a single catheter urine specimen or in a midstream voided urine specimen from a patient whose urethral, suprapubic or condom catheter has been removed within the previous 48 hours.[12] This is as opposed to catheter-associated asymptomatic bacteriuria which is defined as patients with indwelling urethral, suprapubic or intermittent catheterisation with $>10^5$ CFU/ml of >1 bacterial species in a single catheter urine specimen without symptoms compatible with UTI. In patients with spinal cord injury, increased spasticity, autonomic dysreflexia or sense of unease are also compatible with the diagnosis of catheter- associated UTI.[12]

b. Investigations
1. Urinalysis: The minimum investigations for suspected UTI include a urinalysis to evaluate for pyuria and a urine dipstick to evaluate for leukocyte esterase and nitrite. If pyuria is present or if urine dipstick is positive for leukocyte esterase, urine culture should be obtained.
2. Urine culture: $>10^4$ CFU in elderly women with signs and symptoms of UTI, $>10^3$ CFU in elderly men with signs and symptoms of UTI, and also non *E. coli*.

c. Treatment
Treatment should be directed at the organism identified in gram stain and culture. Polymicrobial infections occur in >30% of patients and even more frequently if IDC is used. In these patients, use of a broad-spectrum antibiotic is recommended.

d. Antibiotics precautions in elderly
Of note are three commonly prescribed antibiotics which need to be used with caution in the elderly. Nitrofurantoin is associated with acute nitrofurantoin toxicity and also contraindicated in patients with creatinine clearance <60ml/min. Fluoroquinolones are associated with tendinopathy and tendon rupture. Trimethoprim - Sulfamethoxazole is associated with increased antibiotic resistance in urinary tract infections and also contraindicated in patients with creatinine clearance <15 ml/min.

e. Prevention
Prevention strategies for post-menopausal women include use of non-antimicrobial therapies such as intravaginal oestrogen, oestrogen replacement therapy, and cranberry formulations. However, there is limited evidence to suggest the benefits of cranberry formulations in preventing symptomatic UTI.[10]

Catheter-related UTI causative organisms: *Pseudomonas, Proteus, Providencia, Klebsiella, Enterobacteriaceae, Morganella* and *Enterococcus*. Colonisation occurs in all urinary catheter patients and prophylactic antibiotics are not indicated.

Prevention is key:[12]
1. Catheterise only when absolutely necessary.

2. Remove catheters when no longer needed.
3. Insert catheter using sterile techniques.
4. Anchor catheter to prevent urethral traction.
5. Maintain a closed sterile unobstructed system.
6. Periodically clean the meatus.
7. Observe hand hygiene during catheter care.
8. Consider use of bladder scans to determine if catheterisation is necessary for post-op patients.
9. Consider intermittent catheterisation as an alternative to short-term IDC to reduce catheter-associated bacteriuria and UTI.

Indications for antibiotic management include symptomatic UTI or persistent bacteriuria >48 hours after urinary catheter removal. Urine cultures should be obtained prior to starting antibiotics. Duration of therapy is seven days if patient shows rapid response and 10-14 days if patient shows delayed response, regardless of whether the patient remains catheterised or not. Where possible, remove the urinary catheter. If an in-dwelling catheter has been in place for >2 weeks at the onset of the catheter-associated UTI, and is still indicated, the catheter should be replaced.[12]

f. Treatment of uncomplicated UTI in community dwelling elderly

1. Nitrofurantoin 100mg BD x 5/7; or
2. TMP/ SMX 160/800mg q12H for 3/7; or
3. Augmentin 625mg q8H; or
4. Ciprofloxacin 500mg q12H.

g. Treatment of UTI in catheterised patients

1. IV Gentamicin 5mg/kg OD; or
2. PO Augmentin 625mg q8H; or
3. PO Ciprofloxacin 500mg q12H.

E. Common Skin Infections in Home-bound or Institutionalised Elderly

Skin integrity decreases with age. The skin becomes dry and rough with increased laxity and reduced elasticity. Blood vessels become thinner and more fragile, and senile purpura is often seen. There is loss of subcutaneous tissue, resulting in loss of insulation and mechanical protection.[13,14] Risk factors including trauma; skin wounds; underlying skin lesions, e.g., ulcers, folliculitis, furuncles, fungal dermatoses; neoplasms, e.g., lymphatic cutaneous metastases from neoplasms; extremity stasis or oedema, e.g. peripheral vascular disease, lymphoedema; diabetes; etc.

i. Cellulitis and erysipelas

Cellulitis and erysipelas are common skin infections in the elderly. There is a need to differentiate between erysipelas, abscess and cellulitis. Erysipelas is superficial, involving the dermis and usually sharply demarcated, raised and bright red.[15] It is typically caused by the beta-haemolytic Group A Streptococcus. Cellulitis is an infection involving the dermis and subcutaneous layer. It presents as a faint erythema with rapidly advancing border. The skin may have an "orange-peel" appearance.[15] It is typically caused by *Streptococcus* or *Staph aureus.* Differentials of cellulitis include allergic contact dermatitis and stasis eczema (these tend to be bilateral rather than unilateral), gout and herpes zoster.

Common organisms causing cellulitis include: *Staphylococcus*; beta-haemolytic Group A *Streptococcus*; and group B, C, G *Streptococcus*. Less common organisms are: *Pneumococcus*; and non-group A Streptococcus. In neutropenic patients, infections may be due to *Pseudomonas aeruginosa* or other gram-negative organisms.[15]

Organisms involved in rapidly progressive cellulitis and necrotising fasciitis include *Vibrio vulnificus, Clostridium perfringens, Pasteurella multocida.*

Symptoms: inflamed skin wound that develops rapidly days after injury; local tenderness; warmth; and swelling. May be associated with systemic symptoms including fever, chills, malaise and, in serious infections, confusion and hypotension.

Signs: Rubor, tumor, dolor, calor. Abscess with purulent drainage is the hallmark of *Staphylococcus aureus*. Peau d'orange skin, regional lymph node swelling, necrotic or haemorrhagic bullae (suggests Group A *Streptococcal* cellulitis, *Pseudomonas* spp., *Vibrio* spp., *Clostridium perfringens*).

a. Investigations

Pus drainage and abscess culture in presence of abscesses; blood cultures if systemic symptoms are present; skin biopsy; and fine-needle aspiration are sometimes used. Imaging modalities include ultrasound of soft tissues for *Staphylococcal* infections can be performed in cases of deep-seated infections.[15]

b. Treatment of erysipelas

1. Mild to moderate infections (oral, outpatient management):

-Penicillin V 500mg qds x 7-10/7
-Amoxicillin 500mg tds x 7-10/7
-Cephalexin 500mg qds x 7-10/7

2. Penicillin allergy

-Azithromycin 500mg on Day 1 then 250mg on Days 2-5.
-Clindamycin 300mg qds x 7-10/7

3.Severe infections requiring IV antibiotics

-IV Penicillin G 2 million units q6H
-IV Cefazolin 1gq8H
-IV Clindamycin 600mg q8H
-IV Vancomycin 15mg/kg q12h

c. Treatment for cellulitis (*Streptococcus* and *Staphylococcus* coverage)

In the management of cellulitis, the acronym HAMMER can be used:

H- Hydrate;
A- Analgesia;
M- Monitor fever;
M- Mark off area to watch for progression;
E- Elevate the limb and drain oedema;
R- Record and document the affected site.[16]

1. First-line uncomplicated coverage for *Streptococcus* and MSSA
-PO Cefalexin 500mg qds x 7-10/7
-PO Dicloxacillin 500mg qds x 7-10/7
-PO Augmentin 725mg BD x 7-10/7

Parenteral for more severe infections:
-IV Cefazolin 2g q8H
-IV Vancomycin 15-20mg/kg q12H

2. Second-line complicated refractory or pustular cellulitis for *Streptococcus* and MRSA coverage
-PO Clindamycin 300mg qds x 7-10/7
-PO Linezolid 600mg BD

Parenteral:
- IV Vancomycin 15-20mg/kg q12h
- IV Linezolid 600mg q12H
- IV Clindamycin 600-900mg q8H

Complications of cellulitis include abscesses, fasciitis, septicaemia and osteomyelitis and should be assessed for. If an abscess is present, incision and drainage needs to be performed. If the patient fails to respond to first-line antibiotic, it may be worth considering changing the antibiotic. or extending the course of antibiotic, or admitting the patient for intravenous antibiotics.[16]

d. Treatment for diabetic foot infection
1. Non-limb-threatening:
PO Augmentin 625mg q8H; or
PO Clindamycin 450mg q6H with PO Ciprofloxacin 500mg q12H

2. Limb-threatening:
IV Augmentin 1.2g q8H; or
IV Clindamycin 600mg q6H with IV Aztreonam 2g q8H.

Early debridement to obtain wound cultures is advised to direct antimicrobial therapy. Total duration of antibiotics should be 7-10 days.

e. Necrotising fasciitis
Necrotising fasciitis is dangerous and deep; involving the superficial fascia and/or muscle compartments. They cause major destruction of tissue and is associated with high mortality.[15] It can be monomicrobial (usually involving *Streptococcus pyogenes* or *Staphylococcus aureus* or *Vibrio vulnificus)* or polymicrobial, involving gram-positive and gram-negative bacteria and anaerobes, e.g. *E. coli, Klebsiella* and *P. aeruginosa*.[13,15]

Risk factors for developing necrotising fasciitis include prior injury, such as abrasions, lacerations and surgery sites. Predisposing factors include irradiation, cancer, diabetes mellitus, alcoholism and malnutrition.[13]

A unique clinical feature is that the subcutaneous tissue feels very hard. This is in addition to the features of oedema, skin discoloration or gangrene, anaesthesia or hyperesthesia of involved skin, and usual cellulitis changes.[15] CT or MRI scans may show extension of oedema along the fascia planes. Definitive treatment is fasciiotomy and debridement.

Treatment for necrotising fasciitis:
1. IV Crystalline Penicillin 4 mega units q4H; with
2. IV Clindamycin 900mg q8H; with
3. IV Ceftazidime 2g q8H.

Or:
1. IV Vancomycin 15-20mg /kg q12H; with
2. IV Clindamycin 900mg q8H; with
3. IV Ciprofloxacin 400mg q12H.

If cultures are negative, antibiotics can be oralised to PO Augmentin 625mg q8H.

f. Pressure ulcers
Pressure ulcers are highly preventable.[17] Risk factors for developing pressure ulcers include: (1) immobility; (2) limited ability to sense need for repositioning; (3) malnutrition; (4) faecal and urinary incontinence; and (5) use of restraints.[18] In the elderly, check for (1) skin integrity at pressure points; (2) colour changes; and (3) variations in moisture, heat and firmness.[17] Pressure ulcers can be classified as stage 1, 2, 3, 4 or unstageable. Wounds should be assessed and classified as healable, maintenance or nonhealable; clean or infected.

Preventive measures include use of pressure-relieving devices; patient repositioning; adequate nutrition; good personal care, including moisturising skin adequately; protecting at-risk areas, e.g., xerotic areas, bony prominences; and choosing the appropriate wound dressings for wounds.[13,18] Debridement (chemical, mechanical or biological) may be used to reduce necrotic tissue, bacterial load and promote epithelialisation.[18,19] Topical antibacterials such as silver sulfadiazine, povidone-iodine and topical metronidazole may be impregnated into certain wound dressing products to reduce bacterial load.[18] Systemic antibiotics should be given only if there is clinical evidence of systemic sepsis, advancing cellulitis, or osteomyelitis; otherwise all pressure ulcers are colonised with bacteria.[17] Complications, including cellulitis, sepsis, osteomyelitis, septic arthritis, abscesses and sinus tract formation, should be assessed for. Empiric antibiotics should cover MRSA, anaerobes, enterococci and gram-negative organisms including *Pseudomonas, Proteus* and *Providencia* species.[19]

g. Herpes Zoster
As cell-mediated immunity in the elderly wanes, reactivation

of the *Varicella zoster* virus (VZV) living in the dorsal ganglia occurs. The incidence of herpes zoster doubles in each decade after 50 years of age.[20] The virus migrates to any of the sensory dermatomes, where it erupts as characteristic skin vesicles distributed in a particular dermatomal pattern. VZV reaction is 15x more common in Human Immunodeficiency Virus (HIV) infected persons.[20] Hence the elderly should be tested for HIV when they present with Herpes Zoster infection.[21] Up to 20% of patients with herpes zoster infection develop post-herpetic neuralgia, and this is most common as a person ages.[20]

1. Clinical findings
Prodromal symptoms typically last 1-2 days and include malaise, generalised headache, itching, tingling, burning pain and photophobia with or without fever.[21] This is followed by development of erythematous maculopapular rash which appears in the affected sensory dermatomes and then erupts into painful vesicles and pustules that crust over the next 7-10 days.[20,21] Complete recovery may take up to one month.

Involvement of CN7 can result in facial muscle weakness and zoster oticus which, together, is known as Ramsay Hunt Syndrome. Involvement of the ophthalmic division of the trigeminal nerve can lead to zoster opthalmicus, and Hutchinson's sign (rash on tip of nose) may be a clue.[21] Complications such as blepharoconjunctivitis, uveitis and keratitis may occur and this may result in long-term sequelae, hence patients must be referred for evaluation by an ophthalmologist. Post-herpetic neuralgia is the most common sequelae and is diagnosed when pain persists longer than one to three months after resolution of rash.[20]

2. Diagnosis
In most cases, a clinical diagnosis suffices. The virus is hard to isolate although the infection may be confirmed by giant cells noted on Tzank smear of lesion scrapings. An immunofluorescence assay is used more widely these days.[20,21]

3. Treatment
Antiviral therapy, including famciclovir, valacyclovir and acyclovir, have been shown to reduce the severity and duration of the infection by 1-2 days.[21] They are generally well tolerated. Therapy should be initiated within 72 hours after the appearance of rash. Acyclovir is the "gold standard" of treatment although its use is limited by the inconvenience of the multiple-dosing schedule. The dosage used is 800mg q4H for 7-10/7. Valacyclovir is a safe and effective alternative and given as 1000mg q8H x 1/52. Famciclovir is given 500mg tds x 1/52.
Use of steroids have not been shown to reduce the incidence of post-herpetic neuralgia and its use in combination with antiviral agents is still controversial.[2,20] Recommended dose for prednisone is 30mg BD x 1/52, followed by 15mg BD x 1/52, followed by 7.5mg BD x 1/52 given in tailing dose.

For treatment of post-herpetic neuralgia, for patients with mild to moderate pain, aspirin, paracetamol or NSAIDs such as ibuprofen may be used. To optimise pain control, regular doses of these medications should be prescribed round the clock. Topical medications commonly used to treat post herpetic neuralgia include lidocaine 5% ointment or Capsaicin cream 0.025% to 0.075%. Constant chronic pain can be treated with opioids, tricyclic antidepressants (TCAs), e.g., amitriptyline 25-300mg per day, and anticonvulsants, e.g., gabapentin 300-3600mg per day (taken in three divided doses) or carbamazepine 150-1000mg per day. If pain is persistent and poorly controlled, referral to a pain specialist may be considered. Beware of constipation, nausea, sedation and confusion with opioid use in the elderly and anticholinergic side effects such as dry mouth, drowsiness and constipation with the use of TCAs in the elderly.[21]

4. Prevention of viral reactivation
A live attenuated vaccine, Zostavax, sold by Merck, can be given to adults aged above 60 years old to prevent virus reactivation. It is administered as a one-time subcutaneous injection and has been shown to reduce the overall incidence of shingles in older adults by about 51% and the incidence of post- herpetic neuralgia by 67%. It is safe to be given at the same time as the influenza vaccination.[21]

h. Antimicrobial-resistant organisms (AROs)
Methicillin-resistant *Staphylococcus aureus* (MRSA) is one of the most resistant organisms amongst hospitalised elderly as well as nursing home residents.[22] Generally, there is increased risk for the hospitalised elderly to acquire MRSA as they are usually debilitated with poor functional status, have previously been hospitalised and/ or transferred between hospitals and nursing homes, have multiple comorbidities, have undergone more invasive procedures and are more exposed to antibiotics.[23] A majority of the infections are acquired through contact and hence the simplest, most effective, way to prevent transmission is via hand hygiene.[22] Many of the elderly patients are colonisers. Routine screening for MRSA with cultures taken from specific body sites is carried out for patients admitted to hospitals and nursing homes with a history of AROs, hospitalisation or institutionalisation for 24-48 hours or more within the past six months.[23] The practice of good hand-washing techniques, and wearing gloves when touching blood, body fluids, mucous membranes, non-intact skin and contaminated items, cannot be over-emphasised, even in the home care setting. Gowns and face shields should be worn during activities that generate splashes or sprays of blood and body fluid in homes and nursing homes. Contact precautions, isolation measures (single or cohort room nursing), and decolonisation measures should be in place for patients tested positive.[22]

S. aureus is carried in the nose, throat, axilla, toe webs and perineum of 30-50% of healthy asymptomatic persons.[24] *S. aureus* can colonise wounds and points of entry of any invasive device including intravenous cannula and indwelling catheters. *S. aureus* can cause minor infections such as boils, furunculosis, impetigo, wound infection in pressure sores and venous ulcers, but it can also cause serious infections such as

abscesses, bronchopneumonia, endocarditis and bacteraemia.[24]

Worldwide, antibiotic resistance is a major problem. In patients with *S. aureus* bacteraemia, mortality rate is high from septicaemia and organ failure.[24] Infections caused by MRSA are resistant to Methicillin, Cloxacillin, Flucloxacillin and all other beta-lactam antibiotics.[24] Vancomycin-resistant strains were detected as early as 2002.[24]

1. Screening: PCR testing for MRSA using nasal, groin, axilla swabs.

Decolonisation is needed to protect patients from developing endogenous infections as well as to prevent transmission to other patients by eradicating the pool of infectious cases. Chlorhexidine gluconate 4%, Octenidine dihydrochloride can be used. Hair should be washed daily for 5/7 using antiseptic shampoo and nasal carriage is treated by applying Mupirocin, Chlorhexidine or Neomycin cream. Carriage of MRSA in the nasopharynx is treated with antibacterial mouthwash such as 2% Chlorhexidine gluconate BD and repeat swabs are taken at the end of the decolonisation regime to document clearance.[24]

2. Treatment
Treatment of MRSA infections is with Vancomycin.

i. *Clostridium difficile* infections
Clostridium difficile infection is one of the most common nosocomial infections. It is responsible for 15-25% of cases of antibiotic-associated diarrhoea.[25] Transmission is via contaminated environmental surfaces from infected patients and hand carriage on healthcare workers. The pathogenic strains produce large amounts of exotoxins A and B, resulting in secretory diarrhoea and colonic inflammation. Of all the antibiotics, the Fluoroquinolones, Clindamycins, Ampicillins and 3rd-generation Cephalosporins have been found to have the closest association with *C. difficile*.[25] *C. difficile* infections are largely intestinal and tend to affect patients who are on long-term antibiotic treatment or who are on many antibiotics.

1. Diagnosis
For diagnostic purposes, clinical diagnosis is based on presence of diarrhoea and other associated symptoms, including fever, abdominal pains, general malaise, etc. Colonoscopy may be performed to look for pseudomembranes in the colon although its absence does not rule out the presence of infection.[25] Culture testing is not performed unless for epidemiological purposes. Toxin detection enzyme immunoassays are used instead to identify the presence of toxin A and/or toxin B. The *C. difficile* cytotoxin assay is the gold standard for identification of *C. difficile* infections but is not practical for use in clinical settings.

2. Treatment
Asymptomatic carriers do not need to be treated. Oral metronidazole 400mg q8H for 10-14 days is used, failing which if patient shows no improvement in 72 hours, or if it is poorly tolerated, PO Vancomycin can be used 125mg qds for 10-14 days. Do not use IV Vancomycin as bowel penetration is poor. Recurrent infections can be harder to treat. In severe forms of the infection, where there is severe or bloody diarrhoea, severe abdominal pain, ileus, high fever >38.9º Celsius, TW >20,000/mm3, PO Vancomycin 500mg qds may be used as the first-line drug, sometimes in combination with IV metronidazole 500mg q8H.[26] In complicated cases of toxic megacolon or peritonitis with haemodynamic instability, surgical consultation for subtotal colectomy or a diverting ileostomy with Vancomycin colonic lavage may be needed. Faecal microbial transplantation is the oral or rectal transplantation of faeces from a healthy donor to a patient with recurrent *C. Difficile* infections that have been shown to be efficacious although the precise mechanism is not known.[26] A *C. difficile* toxoid vaccine is currently in the experimental phase of study.[25]

CONCLUSION

Elderly in the home and nursing home are affected more frequently and with more serious consequences by infection. Early identification and treatment is needed to prevent complications of infection and to prevent spread of disease. Identification of the causative organism is useful in most infections to guide antibiotic choice and to prevent spread of antibiotic resistance. Vaccinations remain the mainstay of primary preventive therapy for some infections in the elderly. Pneumococcal and influenza vaccinations should be advocated in this population. Basic infection control practices should be practised in the home settings to prevent the spread of infection.

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LEARNING POINTS

- **Symptoms in elderly with infections tend to be non-specific and subtle. A high index of suspicion is needed because of the associated higher morbidity and mortality in this population.**
- **Common infections in the elderly include bacterial pneumonia, influenza, urinary tract infection, cellulitis, pressure ulcers, herpes zoster, etc.**
- **Assessing pneumonia severity correlates with patient mortality and helps to guide treatment. There are tools that can be used to assess severity.**
- **Most infections are preventable. Preventive therapy with vaccination should be encouraged in the elderly.**
- **Antibiotic resistant organisms are getting increasingly common in the elderly. Discretion in antibiotic use, proper hand hygiene, isolation protocols and environmental sanitation is needed to prevent spread of such organisms.**